

receptor family have been identified. The original member of the family named RAR- α was identified by Giguere et al., Nature 330:624-629 (1987) and Petkovich et al., Nature 330:444-450 (1987). Copies of these publications are filed herewith as Exhibit 1 and Exhibit 2, respectively.

The second member of this family of retinoic acid receptors was identified and cloned by applicants. Initially named *hap*, this retinoic acid receptor is now referred to as RAR- β . (See page 9, lines 6-8.)

Both a mouse and a human homolog of the third member of the retinoic acid receptor family, RAR- γ , have been identified. The mouse RAR- γ was isolated first from a mice embryo cDNA library by Zelent et al., Nature 339:714-717 (1989). A copy of the Zelent et al. article is submitted herewith as Exhibit 3. The human homolog was subsequently isolated and reported by Krust et al., Proc. Natl. Acad. Sci. USA 86:5310-5314 (1989), copy attached as Exhibit 4.

More particularly, applicants' invention relates to the cloned DNA sequence of the RAR- β gene, which is identified in the subject application as the *hap* gene. In the specification and claims, the DNA sequence is characterized by its nucleotide sequence. (See, for example, page 6, lines 1-22.) The retinoic acid receptor encoded by the cloned DNA is related to the steroid/thyroid hormone receptors. (See, for example, page 12, lines 10-12 of the specification.) The RAR- β gene shows tissue specific transcription; the low levels of mRNA are found in most tissues while high levels are found in the prostate and kidney. (See page